MEDICAL WORTHIES OF THE WEST COUNTRY*

BY

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"As there is no man can tell who first contrived the use of houses and clothes to defend us from the injuries of the weather, so the beginning of the art of physick can be no more discovered than the fountain of the River Nile."

THOMAS SYDENHAM, 1624–89

So wrote Thomas Sydenham, sometimes called "The English Hippocrates", and the first notable physician we can claim as a west-countryman. He was born in 1624 at Wynford Eagle in Dorset, in the manor house on an estate bought in the reign of Henry VIII for £40. In a family of ten he was the seventh child, cheerful and happy, but soon to know grief and disaster for shortly after commencing his studies at Oxford, war broke out. As his family were Puritans and the Royalists were preparing to occupy Oxford he returned to Dorset. His father was a captain on the Parliamentary side and four brothers held commissions also. Although he himself was only eighteen he probably served in local engagements. His elder brother Francis was killed in action and even his mother was deliberately shot in a minor action near their village.

It was not until 1646 when he was twenty-two years old that Thomas was able to return to Oxford. There he met with a Dr. Thomas Coxe, whom he describes as the promoter and protector of his early studies. Lectures in those days were on the works of Galen and Hippocrates and from the latter Sydenham learned the value of accurate bedside observation. After less than two years at Wadham College he took his medical degree so that his actual qualifications must have been meagre. He became a Fellow of All Souls and, until his marriage in 1655, was Bursar of the college.

Probably his studies continued during this time but there came another interruption in 1650–51 when Charles II landed in Scotland and Commonwealth forces marched to meet him. In this second war Sydenham served not as a doctor but as a captain of cavalry. Now aged thirty-one, married, with no career, he gave up his fellowship and went to London. For his services he got a grant of £600 but made little headway. Then came the Restoration. He retired to Montpellier for six months' study and finally got a licence to practise in 1663.

How did he differ from his contemporaries? He was devoted to his profession, not so much as a career but as a means of serving mankind. Contrast this with a parody current at the time—" The physicians of the present day are like Balaam's ass; they will not speak until they see an angel." (An "angel" being a ten-shilling piece of that period.)

It was not only his attitude to his profession that was unusual, but also his outlook on disease. He regarded diseases as entities that could be differentiated and classified like plants or animals. He turned medical attention to the scientific study of disease and wrote medical monographs which set the pattern for all time.

There was one disease—gout—on which Sydenham had pretty well the last word. He was a great sufferer himself but it became a blessing in disguise and gave him an opportunity to study it in all its phases so that his treatise on the subject remains the classical description of that disease.

* Presidential address to Bristol Medico-Chirurgical Society, 12th October 1955.

He died in 1689 at the age of sixty-five. His tomb in St. James's, Piccadilly, was practically destroyed in the recent war but the broken stones are being reassembled to remain an inspiration for the future.

THOMAS DOVER, 1662–1742

I would speak next of this pupil of Dr. Sydenham. His connexion was with Bristol, the city which occupies a unique place in the history of the English Poor Law Administration, and Dr. Dover, who will ever be remembered for his famous remedy "Dover's Powder", was the first medical officer of the newly created Bristol Board of Guardians of which I shall have more to say later.

He spent much of his life at sea and I suppose his most famous voyage was to Juan Fernandez, during which he was second captain to Woodes Rogers in the *Duke*, Captain Courtney being in command of the *Duchess*, and William Dampier the Pilot of the South Seas. They left Bristol on 1st August 1708 and sighted the island, after rounding Cape Horn, on 31st January 1709. Many of the crew were sick with scurvy and when Dover put ashore he saw a fire blaze up in the twilight. Thinking a French man-of-war was in the bay he put back to the ships. Next morning, cleared for action, the two ships entered the bay and found it empty. Dover now went ashore again with seven armed men. He soon returned bringing with him a man clothed in goatskins—Alexander Selkirk of Largs in Scotland who had been left on the island four years before by the *Cinque Ports*, a ship he thought unseaworthy. It was he who had lit the fire on the previous evening. I need hardly tell you that this man was the prototype for Robinson Crusoe in Daniel Defoe's famous book published in 1719. We are told that during his stay he killed 500 goats and lived largely on their flesh with crawfish, turnips and cabbage from the cabbage-trees. "On first coming on board his speech was almost unintelligible from long disuse for he seemed to speak his words by halves."

Dampier had been with Selkirk on the *Cinque Ports* and recommended him so strongly that he was at once appointed mate of the *Duke*. Next day tents were set up on shore for the sick men. Selkirk caught two or three goats each day and the scurvy patients rapidly recovered on a diet of goat's meat, turnip tops and watercress. They sailed on 13th February north along the coasts of Chile and Peru to begin the real business of the expedition—the raiding of the Spanish coast. They captured many ships and finally arrived back in England on 14th October 1711 after a voyage of three years and two months.

£14,000 was spent on fitting out the ships but the net profits were at least £170,000. Dover must have received several thousand pounds as his captain's share besides dividends as an owner. As chief medical officer he received £423. Amongst his treasures were two fine silver candlesticks which he sold to the Town Clerk of Bristol, John Romsey, for £114. Romsey presented them to Bristol Cathedral in 1712 and I believe they are still used on the altar there.

An original copy of his book *The Ancient Physician's Legacy to his Country* can still be seen in the University Medical Library.

Thomas Dover was an interesting character, part doctor, part sea captain, part adventurer and possibly part pirate. He achieved a full and adventurous life. On more than one occasion he sailed from Bristol in charge of a ship on some pioneering expedition. His greatest claim to medical fame must be his powder which is still possibly one of the most effective cures for influenza and even the common cold, which, with all our vaunted knowledge, we doctors have not yet conquered. He died on 20th April 1742, aged eighty years and was buried most probably in the vault of the Tracey family at Stanway, a village about twelve miles north-east of Cheltenham but no memorial now remains.

THE BRISTOL BOARD OF GUARDIANS

The author of this scheme was a Mr. John Cary, a resident in the city and churchwarden of St. John the Baptist in 1679 and 1680. An act establishing in Bristol the

first Board of Guardians was introduced into Parliament in 1696. This authorized the provision of a workhouse and the raising, by a rate from six parishes, of funds to provide maintenance for the destitute poor. The main features of that scheme remained until quite recent times for the Board of Guardians continued until 1st April 1930 when as a result of the Health Act of the previous year such boards were superseded by Public Assistance and more recently Social Welfare services.

The Board consisted of the Mayor, the Aldermen and forty-eight Guardians "to be chosen out of the honestest and discreetest inhabitants of the said City". The first election was held on 12th May 1696 and the Guardians set to work with great energy to carry out the provisions of the Act. They assumed semi-magisterial duties and had strangers and disorderly persons apprehended and committed to "Bridewell" for various periods. The keepers of disorderly houses were punished and the Corporation set up a pair of stocks and a whipping post, whilst chains and bolts to fasten disorderly persons to blocks, were provided in a portion of St. Peter's Hospital known as "purgatory". This building, the old mint in Peter Street, was acquired in 1698 for £500 and survived until the disastrous air raid on 24th November 1940 when this exquisite gabled building was utterly destroyed. It was the headquarters of administration for many years and as the boundaries of Bristol grew so the work increased. The Seal of the Bristol Board of Guardians was a most appropriate one. It consisted of a picture of the dome-shaped straw type of beehive, the motto beneath being "Hyemis memores laborant".

At this point I must say a few words about Southmead Hospital.

SOUTHMEAD HOSPITAL

By an Act of Parliament in 1897 the Bristol Corporation obtained the transfer of the parishes of Stapleton and Saint George and part of Horfield from the Barton Regis Union and also parts of Bedminster and Keynsham Unions, combining the whole of the extended City into one Poor Law Union. It therefore became necessary on 1st April 1898 for the Barton Regis Guardians to provide a new workhouse for the smaller part of the work that remained to them. They acquired thirteen and a half acres of land at Southmead, Westbury-on-Trym, for the erection of this new building at a total cost of £33,000. The foundation stone was laid by the Dowager Duchess of Beaufort on 18th September 1900 and the building was finally declared open on 18th September 1902 by the Rt. Hon. Sir John Dorrington, Bart., M.P.

A further extension of city boundaries in 1912 ultimately engulfed the new infirmary. Barton Regis ceased to exist as a Board and in taking it over the Bristol Guardians decided on a very progressive policy for those days. They enlarged Southmead at an additional cost of £44,000, building modern wards capable of accommodating about 650 "chronic sick" patients.

These new wards were completed about August 1914 and immediately the Bristol Board offered them as a War Hospital. Thus Southmead became part of the 2nd Southern General Hospital for the reception of war casualties. During this first Great War a new operating theatre was added and during the war years upwards of 17,000 wounded were treated in the hospital. The War Office retained control until October 1920 when the hospital was handed back to the Guardians.

Its first Medical Superintendent under civil control was the late Dr. Robert H. Norgate who died in December 1924. My own connexion with Southmead Hospital began in February 1923 when I was appointed assistant medical officer, ultimately being elected Medical Superintendent in April 1925.

With the passing of the Poor Law régime as a result of the Health Act of 1929, Southmead now passed to the control of the Health Committee of the City of Bristol, being taken over on 1st April 1930. Gradually its scope was increased and in the Second World War it took its full share of the casualties arising from the disastrous air raids from 1940 onwards. However, it is interesting to note that the number of these (635) in no way compares with the many thousands admitted during the war of 1914–18.

Since 1948, Southmead Hospital has been further developed to become a large general hospital with the largest maternity department in the whole of the south-west.

THOMAS BEDDOES AND THE MEDICAL PNEUMATIC INSTITUTE

Thomas Beddoes, our next character, founded the Medical Pneumatic Institute in Bristol in 1793 in Dowry Square, Hotwells. Here diseases of the lung, such as tuberculosis, were treated by the inhalation of oxygen, hydrogen and carbon dioxide. Beddoes has himself been largely forgotten but his colleague and assistant are both remembered.

James Watt the engineer was the colleague, Humphry Davy, the assistant. In 1800 Davy published the result of his work on nitrous oxide which soon became known as "laughing gas". He wrote "as nitrous oxide in its extensive operation appears capable of destroying physical pain, it may probably be used with advantage in surgical operations."

Unfortunately, little attention was paid to Davy's work. Laughing gas was too funny to be taken seriously!

Literary men of the time, Wordsworth from Alfoxden, near Holford, Southey and Coleridge came to Beddoes' Pneumatic Institute, not to be cured of any disease but to seek the exhiliarating effects of doses of the new gas. Nitrous oxide is still used extensively with oxygen or air as one of the safest anaesthetics. It brings relief to countless thousands in childbirth; it is the only anaesthetic which a midwife is permitted to use on her own responsibility—in a special machine called "Minnitt's apparatus". Had Davy's work and wisdom been followed up, anaesthetics would have been available at least forty-six years earlier, for it was not until 1846 that ether and later chloroform first came into use.

To digress for a moment, James Young Simpson in Edinburgh first used ether successfully on 19th January 1847 in delivering a woman of her second child. In March, he published a paper recording the use of ether in several cases.

Chloroform was discovered in 1831 by Liebig in Germany. It was first used by Simpson in November 1847. Then commenced the battle over chloroform. Dublin contended that it would increase the incidence of bleeding, convulsions, paralysis, pneumonia and so on. Simpson indignantly flourished figures over the heads of his critics showing that shock and exhaustion were much greater dangers than chloroform.

Then the moralists joined the fight and even brought religious objections. Simpson's opponents took their stand on Genesis iii. 16: "Unto the woman he said, I will greatly multiply thy sorrow and thy conception; in sorrow thou shalt bring forth children." Simpson, well versed in the Bible, soon countered this with the first Biblical operation (Genesis ii. 21): "And the Lord God caused a deep sleep to fall upon Adam and he slept; and He took one of his ribs and closed up the flesh instead thereof." Before the clergy had time to work this out, he published a famous pamphlet: *Answer to Religious Objections advanced against the Employment of Anaesthetic Agents in Midwifery and Surgery*.

This was headed by the texts: "For every creature of God is good, and nothing is to be rejected if it be received with thanksgiving." (1 Timothy iv. 4.) "Therefore to him that knoweth to do good and doeth it not, to him it is sin." (James iv. 17.)

Medical, moral and religious objections were swept aside and more and more chloroform was used. The most progressive lady of the time—good Queen Victoria—dealt the final blow, for in April 1853 Prince Leopold was born to Her Majesty whilst she was under the influence of chloroform!

Her physician, Sir James Clark, wrote to Simpson to his great delight: "The Queen had chloroform exhibited to her during her recent confinement. It was not at any time given so strongly as to render the Queen insensible, and an ounce of chloroform was scarcely consumed during the whole time. Her Majesty was greatly pleased with the effect and she certainly never had a better recovery." I like to think that Bristol and the West Country had a share in this romantic battle against pain, and to Thomas Beddoes should go some of the credit for his initiative.

EDWARD JENNER, 1749–1823

This most famous of all general practitioners is the most illustrious name I can think of associated with the West Country. He lived through the turmoil of the Napoleonic Wars, when the whole of the civilized world was ravaged by a war of almost unequalled ferocity. Before he died, he had the supreme satisfaction of knowing that his discovery had been the means of saving more millions of lives than had been sacrificed during that conflict. Before his time, no one had conquered a disease and most treatments were directed to the relief of symptoms.

The disease he conquered was smallpox. Nowadays the fear of smallpox has been so completely banished that it is difficult to realize what a haunting dread it once was. However, in the *Gloucester Journal* of 23rd February 1823 we read in an obituary notice of Dr. Jenner that 40,000 people died annually of smallpox in the British Isles, that this disease killed one in fourteen of all who were born and one in six of all who contracted it in a "natural way". Nor was the fear of death all; there was also the dread of disfigurement.

Jenner's early life was interesting. At the age of twenty-one, after serving his apprenticeship to a surgeon at Sodbury, near Bristol, young Jenner went to London and lived for two years as a house pupil of John Hunter. This was the beginning of a lifelong friendship between these two. Jenner learned from Hunter's maxim "Don't think, but try; be patient, be accurate."

During his apprenticeship, a young dairymaid had said to him, "Oh! I shall never have smallpox, for I've had cowpox." When Jenner settled in Berkeley in Gloucestershire, he soon found that all the country folk held firmly to this belief whilst all the local doctors laughed at it as an old wives' tale, but Jenner was patient. For twenty-five years, he studied the affections of cow's teats and udders. He found true cowpox rare as compared with the many other conditions simulating it, which were sometimes conveyed from the cows to the hands of the milkers. These spurious diseases did not protect but true cowpox, once sustained, did. At length, the time came for an experimental trial. On 14th May 1796, this undistinguished country doctor successfully inoculated a healthy lad of eight years with cowpox, using secretions from the lesions on the hands of a milkmaid. Sarah Nelmes was the milkmaid and James Phipps the small boy. Eight weeks later, Jenner inoculated Phipps with true smallpox. Nothing happened! The cowpox had prevented the boy from contracting the fell disease.

Two years later, Jenner wrote a book of seventy-five pages entitled *An Inquiry into the Cause and Effects of* Variolae Vaccinae, *a Disease discovered in some of the Western Counties of England, particularly Gloucestershire, and known by the name of Cowpox.* Encouraged by students he had met at John Hunter's, Jenner went to London. At first there was opposition but gradually more and more physicians and surgeons tried. The idea was gradually accepted in England, then in Europe and finally America. He was honoured by Spain, Italy and Denmark, and the Empress of Russia presented him with a diamond ring. At the outbreak of war between Britain and France many Englishmen living in France were held prisoner by Napoleon. All the usual diplomatic requests failed to secure their release. Jenner then wrote direct to Napoleon and the Emperor immediately ordered the release of all prisoners, saying "We can refuse nothing to that man." Fame and reputation were his, but great patients and large fees never came his way. Vaccination was a simple procedure which anyone could perform. Jenner went back to his country practice and worked quietly on till in his seventy-third year he died of apoplexy.

WILLIAM GILBERT GRACE

Grace was born at Downend, near Bristol, in 1848 and by common consent was the greatest cricketer the game has ever known. During his career of nearly forty years, he twelve times headed the list of batting averages and compiled over 200 "centuries". As a bowler, he was only slightly less successful and when over fifty years of age he still retained a commanding position in first-class cricket. His first great match was in

1864 when he played for South Wales at Kennington Oval, and immediately afterwards he made 170 at Brighton against the Gentlemen of Sussex. In 1868, he scored 100 twice in one match—an achievement that he repeated on three occasions. Engaged in first-class cricket for thirty-six years, he fairly earned the title of Champion. His highest score of 400 not out was made against Twenty-two of Grimsby in 1876. His highest score in first-class cricket was 344. In 1875, he took 192 wickets in one season. Playing against Oxford in 1886, he took all the wickets in one innings for 49 runs. In 1899, he became secretary and general manager of the London County Cricket Club at Crystal Palace. He was largely responsible for establishing cricket as a national pastime as we now know it.

Medical men are always proud to claim him as a member of the profession and I have two original stories to tell you, which I think have not been told before. Both show his sterling human qualities and his skill as a doctor. Dr. Grace was very friendly with another cricketing character, Sammy Woods, and these two often trained together for great occasions. Part of their training consisted in long walks over the Mendip Hills. After such an expedition one very hot summer's day, W. G. became very thirsty and suddenly exclaimed to his companion "What wouldn't I give for a drink of beer, Sammy!" Without answering, his companion disappeared over a stone wall and in a few minutes returned pulling from his pockets two bottles of beer. Grace gazed in amazement at this apparently magical feat. He could only stammer "Where in the world did you get those?" To which Sammy gaily replied, "Oh! that's easy. I've got 'em planted all over these Mendips."

In 1937, at the end of a meeting of a women's guild at which I had been speaking, a very old lady came to me, very proud to tell her story of Dr. W. G. Grace.

It seems that as a young woman she had lived in a very poor part of the city, near Quaker's Friars. Her own doctor, who charged the extraordinary fee of five shillings for a confinement, was apparently very good but rather addicted to the bottle. So much so, that when her time came and her husband went to get the doctor, the latter's wife could only say, "I'm sorry, Mr. So-and-So, but my husband is in no fit state to come." The man, taken aback, was quickly comforted when he was told to go and find Dr. Grace. This meant a considerable journey, late at night but away the man went. Although W. G. was already in bed, he quickly got up and dressed, got out his "dog cart" and with the anxious husband up beside him, drove to the patient's house.

My old lady was apparently better tended than she had ever been before. However, the point that remained so clearly in her mind was that as soon as Dr. Grace saw her husband he said, "Have you any eggs and brandy in the house? She'll want them tomorrow." The man could only dejectedly shake his head. He had none and also lacked the means to get either. But the generous-hearted W. G. was equal to the occasion. Patting the husband on the shoulder, he said, "You come round to my house in the morning and I'll give you some eggs and half a bottle of brandy."

A truly great cricketer, a kindly doctor and a gallant gentleman.

ERNEST WILLIAM HEY GROVES, 1872–1944

Almost eleven years ago—on 22nd October 1944—Ernest Hey Groves died in Clifton. It is with some diffidence that I mention one who was so recently with us but I do so because I was his surgical dresser and have a vivid recollection of his sterling qualities, both as a surgeon and as a man. Finally, it fell to my lot to assist in caring for him during his long and distressing last illness, during a great part of which he was nursed in Southmead Hospital.

Groves was born in India in 1872 and was the son of a Civil Engineer who settled in Bristol on his retirement, when the boy was only three years old. He went to school in Bristol, gained an entrance scholarship at Bart's, took B.Sc. in London in 1890 and qualified M.R.C.S., L.R.C.P. in 1895. He took his M.B. in 1897 and M.D. in 1900. After being Resident Accoucheur at Bart's under Sir Francis Champneys, he was in general practice in Chewton Mendip. He once said to me "Many is the time I've

wandered over those Mendips searching for a hypothetical half-crown." Later, he settled in Kingswood, where he carried out many surgical operations in his own home, and where his wife, who had been a sister in St. Bartholomew's Hospital, helped by nursing the patients. In 1905, he gained high academic honours by taking the London M.S. with Gold Medal and the F.R.C.S., a truly remarkable record for a man engaged in practice.

In the war which came soon afterwards, he went to Egypt in charge of the Surgical Division of a General Hospital and there elaborated his extension splints for fractures of the lower limb and became one of the pioneers in orthopaedic surgery. He was most friendly with Sir Robert Jones who visited him many times in Bristol, thus cementing their common interest in bone surgery.

Perhaps Hey Groves's greatest triumph was the production of the *British Journal of Surgery* and of which he was Editorial Secretary for more than twenty-five years. The *Journal* was founded in 1913, and it was largely through his enthusiasm and industry that it has attained a high position in the world as a surgical publication.

In the very early twenties he, together with the late Dr. Carey Coombs, did his utmost to promote harmony and co-operation between the General Hospital and the Royal Infirmary in Bristol. Amalgamation was much in the air in those days, but the plan misfired and was delayed for over twenty years.

As a surgeon, no one could be bolder when the occasion demanded. The most dramatic operation I can recall was about 1921, when tumours of the lung were rarer than they are today. A Swedish sailor had been found to have such a tumour in the left chest. Of course, the question of aneurysm had been considered but this seemed too big for such a diagnosis, and finally physicians agreed that biopsy was the only answer. At least fifty persons crowded into the theatre to see this unusual sight. The tumour was displayed by a wide incision and a trap door of ribs raised and a section taken, the report being returned by the pathologist a few days later as "laminated clot"! However, the patient survived for several months before dying of a sudden haemoptysis.

From 1922–32, Hey Groves was Professor of Surgery and his work was recognized when the University conferred upon him an Hon. D.Sc. at the Centenary of the Bristol Medical School in 1933.

His *Synopsis of Surgery* is well known and he published many other books for students and nurses besides being a prolific writer in the journals.

He was a good billiard player and a fine swimmer, especially under water. He could dive in off the highest board at Henleaze Lake and amaze everyone by the length of time that elapsed before he "surfaced" again. As a host and companion, he was unsurpassed. He loved the novelty of travelling and visited Australia for the B.M.A. meeting in 1935, being President of the Orthopaedic Section on that occasion.

The final phase of his work came when he was group officer for Bristol in the E.M.S. at the outbreak of the Second World War. All his old enthusiasm was revived at this time and it was a shock to his many friends and colleagues when he collapsed from a stroke which rendered him helpless for so many months. So passed a great English surgeon whose name is inseparably connected with Bristol and the West.

THE GROWTH OF HUMAN TUMOURS IN LABORATORY ANIMALS

A new approach to the Assay of Anti-Carcinogens

BY

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Although metastatic breast cancer is at present beyond surgical cure the disease can be palliated by removal of the ovaries and adrenal glands. The first adrenalectomy was performed by Charles Huggins of Chicago in 1951. After operation the secondaries in some of the cases rapidly melt away but this effect is unfortunately only temporary.

One advantage of this type of clinical treatment however is that we have learned indirectly how cancer can be influenced by altering the body tissues in which the tumour grows.

Another line of attack on this problem is that of transplanting human tumours into laboratory animals. This provides a means of studying the effects of certain chemicals, hormones and viruses on human breast cancer outside the patient and gives a laboratory testing-ground for them before trial on patients.

This paper describes briefly how human tumours can be grown in rats and hamsters for this purpose. The method is not new for in 1913 Carrel and Burrows working in this country showed that human tumours could be grown when implanted into animals. More recently Dr. H. S. N. Greene of Yale University and Dr. H. Toolan of the Memorial Center in New York have revived and developed this method. They have found that transplants will grow when inoculated into the brain, anterior chamber of eye, the subcutaneous tissues of the rat or the cheek pouch of the hamster.

The technique of the cheek-pouch implant in the hamster is very simple. The animal is anaesthetized and a piece of tumour the size of a large pin's head is introduced into the cheek pouch through a small puncture wound. Plate VI (*a*) shows the tumour growth from an implant after eighteen days.

Subcutaneous implants are carried out in rats. A fine suspension of minced tumour in a buffered solution is injected into the subcutaneous tissues of one flank through a wide bore needle and syringe. Plate VI (*b*) shows one of the implants growing in rat.

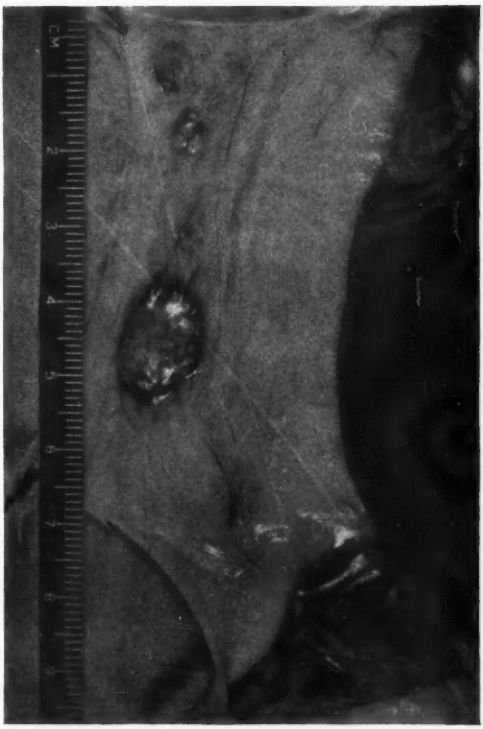
For the tumour to grow in the animal it has to obtain a new supporting stoma and blood supply locally from the rat's tissues. At this stage the unprotected cancer cells of the implant are highly vulnerable to the host reaction. For the implant to survive this the animal's tissue reaction has to be temporarily modified. This can be effected by giving small doses of cortisone, X-rays or a combination of the two.

Human cancer grown by these means in animals can, once it is established, be used as a method of testing anti-cancer substances. Subcutaneous or submucous tumours are easily accessible to direct palpation to assess changes in size. Direct measurement of changes can also be made with a pair of calipers.

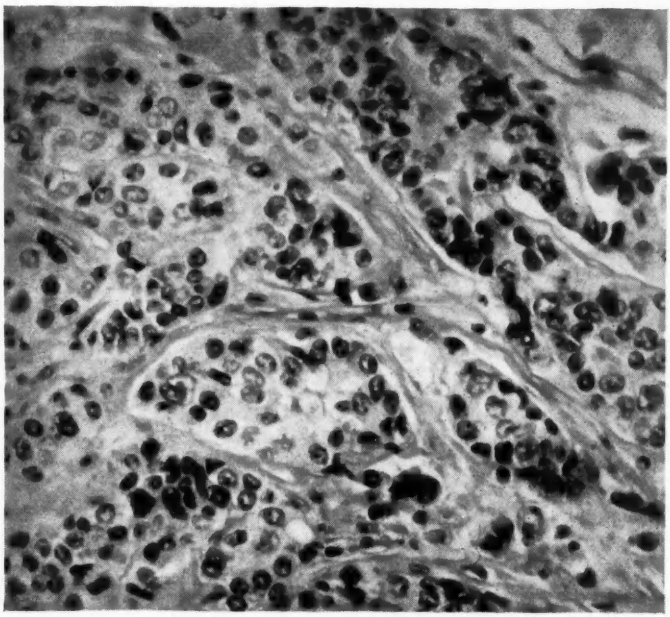
One criticism of this method has been that these human tumours growing in animals lose their human characters and become an animal tumour. This has been nullified by some recent work carried out at the Memorial Center in New York. A specimen of highly anaplastic cancer of the cervix was obtained from an inoperable case. This was grown in rats and hamsters on serial transplantation by the methods previously described. Later with the patient's co-operation and wish it was re-transplanted beneath the skin of the forearm. Two weeks later the transplant was excised and by that time it was again growing well. This seems to abolish the criticism that these human tumours grown in animals lose their human character.

PLATE VI

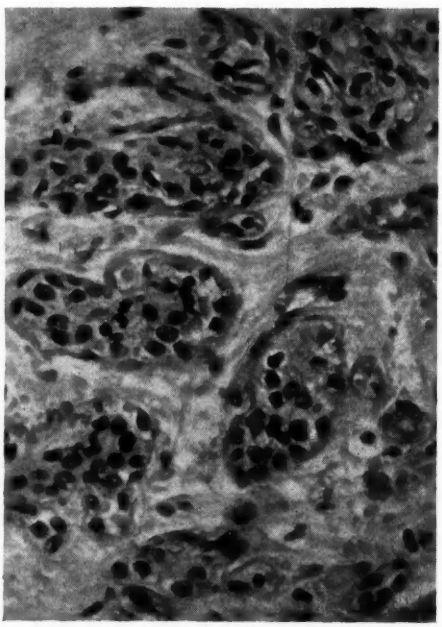
(*a*) *Human breast cancer implants in hamster cheek pouches, 18 days after inoculation.*



(*b*) *Human breast cancer implant injected below skin of rat flank, 14 days growth. (Note seedling growths along needle track and that the tumour is obtaining a blood supply from the host.)*



Original tumour (× 240).



Rat flank implant (× 240). *Third-generation specimen, i.e. growing 42 days after removal from patient.*

This technique also provides an easy method of observing the behaviour of cancer cells in normal and cancerous animals. It provides a method of studying the growth and spread of cancer and the question of resistance and immunity to the tumour process.

Plate VII is a histological specimen of human breast cancer growing in the rat's flank. The original, shown with it for comparison came from a primary cancer of the breast removed by radical mastectomy.

(This work was carried out during the tenure of a British Empire Cancer Campaign Fellowship at the Memorial Center, New York.)

DOMESTIC DEATHS FROM ELECTROCUTION

BY

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Every year about a hundred people in England and Wales lose their lives by electrocution. Table I shows the total number of accidental deaths during the years 1942 to 1953, whilst Table II shows the number of deaths in 1952 and 1953 divided into those at work and those in the home. It is these deaths in the home that are considered in this paper.

When the wiring is done by an expert accidents are rare and most deaths are due to the ignorance and carelessness of the householder. Too often there is evidence of unsatisfactory wiring carried out by an amateur to effect replacements or improvements. Other common causes of accidents are neglect to obtain competent advice following " little shocks " from appliances, failure to repair damaged or worn insulation and attempts to carry out repairs without turning off the main switch.

The ordinary domestic electricity supply in England is at a pressure of about 240 volts, a figure which was agreed in 1946 and has been mandatory since 1947. Its strength, which is dependent upon the resistance between the terminals, will vary from 5 amperes for lighting to 30 amperes for heating and cooking points. Alternating current (A.C.) has almost completely replaced direct current (D.C.) and the standard is usually of 50 cycles per second. As a legacy of the past there are still in operation systems with non standard pressures ranging from 104–250 volts (A.C.) and others which operate at voltages of 100–240 volts (D.C.). The minimum current to kill from electrocution is stated to be about 65 milliamperes.

Death from electricity can occur in two ways, either by a sudden surprise shock causing vagal inhibition or by true electrocution which produces ventricular fibrillation or respiratory failure. Although the importance of the element of surprise should not be over emphasized, cases have been recorded in which death has occurred from touching a wire which was believed to be live but which was, in fact, dead.

From a practical point of view, it should be stressed that to receive the full effect it is necessary for a good " contact " to exist with " live " point and with " earth ". Dry human skin is a poor conductor with an average resistance of 2000–3000 ohms but when it is wet the skin resistance is reduced to as little as 500 ohms. If the skin is punctured the effective resistance will be that of the subcutaneous tissue, which is always very much lower than the skin. A current of about 12 milliamperes may make it impossible for a person to release his grip upon the " live object " if adequate " earth " is present. Other modifying factors will be the duration of exposure and the area of contact, whilst it is possible that the actual voltage received may be less than that computed because of electrical leakage.

It is a popular idea that water is a good conductor and therefore that a person is more vulnerable to electrocution when immersed in a bath. This is not always the case, for during the investigation of the case of R. *v.* Whybrow, which is mentioned later, experiments showed that a person sitting in that particular bath was, in fact, insulated. The bath itself is not the true reason for the bathroom being one of the two most " electrically " dangerous rooms in the house (the other is the kitchen) for the risk lies in the damp atmosphere when associated with a large number of metal objects

(mostly well "earthed") such as the water taps and pipes, gas pipes and the like. For this reason it is necessary either to instal the electric switches outside the bathroom, or, if inside, to operate them with a pendant pull cord of non-conductive material. In spite of this safety precautions are rendered quite valueless by the introduction of some portable electrical apparatus such as an electric fire, a hair dryer, an electric kettle or even a wireless set. All, if properly constructed and intelligently used are safe enough but unfortunately they have often been made unsafe by unskilled wiring or rough handling. It is therefore no great surprise to hear of deaths from electrocution from time to time caused by foolhardy handling of electric appliances when seated in a bath.

When accidents occur, it is not uncommon for the electrical expert to find that an electric kettle or heater has been amateurishly rewired with omission of an earth lead (3rd wire) which makes it inevitable that, in the presence of a fault rendering the casing alive, any person handling it will take the full force of the current.

One early morning a husband heard a thud in the kitchen to which his wife had gone to make an early cup of tea. He got up at once and found his wife lying dead upon the kitchen floor. She had apparently been electrocuted from the casing of an electric kettle. This was confirmed by electric burns on the thumb and finger, whilst earth had occurred from her arm being in contact with one of the metal objects in the kitchen. The plug was examined by an expert electrician who found:

(1) There was no earth wire connected.

(2) That the person who had wired up the plug had left sufficient loose wire to flap over and short on to case. (Fig. 1).

Even a properly wired kettle may be dangerous if handled by an ignorant person as the following case will illustrate:

An elderly woman went out of her house one afternoon and forgot to switch off her electric kettle. This boiled dry during her absence and the safety mechanism blew out the plug making it impossible for her to replace the plug after refilling the kettle. Under the impression that there was "something stuck" she took the plug in one hand, thereby earthing herself with the earth safety metal strip upon it, and with the other hand introduced a metal screw driver into the positive terminal which was still connected "to a 25-ampere power plug at 'on', she somewhat naturally received the full charge with fatal results".

Careless maintenance of electrical equipment may well result in death and it is most surprising that many housewives of more than average intelligence will continue to use vacuum cleaners whose flex is bare from constant wear and tear. Recently a similar accident occurred with an electric lawn mower.

Two other kinds of domestic electrical equipment deserve mention—the radio and television sets. The former have been the cause of fatalities and afford an excellent example of the casual manner adopted by the public towards electricity. How often has a person been heard to say "A funny thing happened this morning, I got a shock off my radio" and when asked what had been done about it given the reply "Nothing"?

Fatal accidents have been recorded from television sets, in one case due to the "frame" coming in contact with a metal grill in front. The fault in this particular set was completely remedied.* Nevertheless, education of the public to take notice of small "shocks" and call in an expert electrician would pay dividends.

Children, having small fingers, may be electrocuted by "poking" them into the holes of the standard plug or alternatively using metal objects such as skewers or knives. The close proximity of excellent "earths" such as gas pipes makes the "amusement" even more dangerous. This risk can be reduced by fitting plugs with safety shutters. A somewhat similar type of accident occurred some years ago under the following circumstances.

* The company called in all sets and modified them.

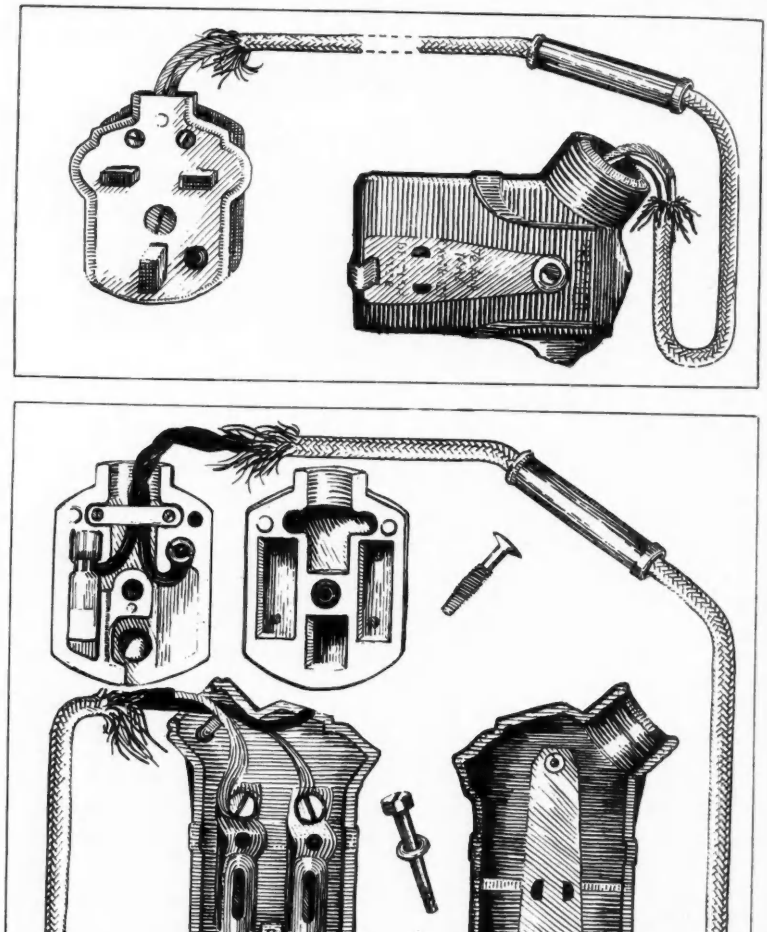

FIG. 1

Above: Kettle plug as found (note frayed wire and cover out of place—no earth wire).
Below: After dismantling (note bare loose wires).

A child aged five years was playing in a hall closet. Suddenly it collapsed and a doctor who was called found it to be dead. Detailed examination showed electric burns on the tips of the right index and middle fingers, one of which had a round depressed area. The post-mortem findings internally were consistent with electrocution. Examination of the closet showed a porcelain fuse box with bare wires sunk into the top in gutters too small to allow access of an adult finger but large enough to admit that of a child's finger. The bare end of the wire exactly fitted the depression on the child's finger and evidence showed that the child had been standing bare-footed upon a gas pipe when collapse took place.

Other fatal accidents have occurred from children climbing up and touching overhead electric cables and on one occasion a boy scaled a pylon for the purpose of throwing two metal cups tied together with string over the cables carrying the grid across the Thames. A blinding flash was seen for some miles away and the body of the culprit was found at the base of the pylon with a " broken neck " and extensive electrical flash burns down the whole of one side of his body resembling crocodile skin.

A case was recently recorded in the press of a child electrocuting itself by chewing through a length of electric flex.

EVIDENCE OF ELECTROCUTION

It has been already stated that deaths from electrocution may be due either to ventricular fibrillation or respiratory failure, unfortunately the post-mortem appearances of either are not peculiar to the effect of passage of an electric current, for they are quite commonly seen, for example, in deaths resulting from coronary insufficiency or cerebro-vascular catastrophes.

The proof that electrocution is the cause of death must depend upon the identification of an " electric " burn. This demands a careful scrutiny of the body surface of *everyone* who dies in close proximity to any electric "live point".

The appearance of an electric burn has been *described* as a hard blister with a central white parchment bleached area and a hyperaemic zone of surrounding skin. The identification and interpretation of this lesion may not be easy and there is also considerable doubt as to whether such an appearance may not also be produced by exposure to an electric current shortly after death. The actual appearance of the electric lesions will vary according to the firmness of the contact. Thus, if the contact is poor in pressure and area, there will be small pricked out " spark burns ". If a diffuse area is involved with moderately firm application there may be a shaped burn which after a few hours may even show charring, especially with high voltage currents. The exit lesion may be less obvious and not so characteristic. In true electrocution burns may be completely absent, and it should be borne in mind that this may occur where a person is grasping an object, such as a pistol grip drill, at the time it becomes live. Electric burns are usually more extensive than their appearance suggests, and later considerable sloughing may occur.

Histologically an appearance has been described of elongation of the basal cells and nuclei but unfortunately this is by no means consistent and can occur from thermal burns.

The two alternative forms of death have already been mentioned. Probably most domestic deaths are due to ventricular fibrillation and the post-mortem appearances closely resemble those due to coronary artery insufficiency. Those due to respiratory failure will show more pulmonary oedema and certainly occur when the current passes through the medulla oblongata.

Suicide by electrocution is uncommon and such cases as occur are, as might be expected, somewhat bizarre in character. On one occasion death was brought about by preparing a circuit from the domestic supply using rings on a finger of each hand

to connect to a plug, whilst in another a complicated electrode was placed over the praecordium, the circuit being closed by a wire from the same plug being held in one hand.

The use of electricity for homicide is believed to be very rare but it may be that cases have passed unrecognized owing to lack of identification or even search for electric burns. The following case illustrates an attempt at murder and scrutiny of it will show how easily it could have been missed if the woman had died and no suspicion had existed.

R. *v.* WHYBROW (*Essex Assizes* 1952)

In this case the accused was a married man living with his wife and children near Southend-on-Sea. One evening his wife decided to take a bath and whilst she was doing this he was in a bedroom on the other side of the passage. Suddenly she cried out saying that she had received an electric shock when she had touched the soap dish. After a short interval he went to the bathroom and decrying the suggestion of a shock touched the soap dish to show that she was mistaken. At the time she was satisfied with his statement but the next morning decided to telephone the Electricity Company to ask them to call and examine the soap dish. They were too busy to do this immediately and consequently she decided to examine it herself with the aid of her sister-in-law. After scraping away the plaster she found that it was fixed to the wall and that there was a wire to the bracket which had been made from a piece of tin by her husband. She traced the wire under the bath (boxed in) through the wall into the adjoining lavatory, where, after winding around the base of the lavatory pedestal, it entered the roof space above and then ran across the top to a cupboard in the bedroom where it was connected to a metal tube secured between two pieces of bakelite which were screwed to the wall. She then called the police who carried out a search and in a drawer found a length of india-rubber covered electric wire one end of which was attached to the positive terminal of a 15 ampere plug and the other to a large split pin which exactly fitted the metal tube in the cupboard. In the circuit had been introduced a 15-watt bulb with holder. (Fig. 2). A 15-ampere power-plug was fixed on the skirting board adjoining the cupboard and the whole circuit when connected allowed a current of 65 milliamperes to be detected at the soap dish (the reduction was due to the resistance of the bulb). Examination of the bath showed that a person seated in it would be completely insulated. This was due to the porcelain being intact, the runaway being insulated by the plaster which fixed it, leaving the only "earth" through the chain attached to the bath plug. An appreciable electric shock could be felt when the soap dish was touched.

Investigation showed that the husband was associating with a young girl without telling her parents that he was already married. His wife had discovered and resented this liaison. He had moreover been previously convicted of bigamy. His defence was that the apparatus was the earth of his wireless set but the jury found him guilty of attempted murder.

To summarize, the recognition of death from electrocution is not easy unless the medical practitioner who first sees the case is both suspicious in his approach and careful in his examination of the body. Two fatal cases of electrocution, although the possibility was considered and discarded at the time, were not detected until some weeks later. In neither case was there anything to suggest electrocution shown by autopsy and no local burns were present.

Examination of cases of electrocution which have occurred each year shows the main reason for the accidents is faulty wiring installed by amateurs.

The medical literature on electrocution is not extensive but valuable information on the subject can be found in the technical journals. The electricity authorities are always most courteous and helpful, and in this connexion I should like to express my thanks in particular to Mr. L. H. Jesty of the London Electricity Board for his helpful advice both in the preparation of this paper and in connexion with several cases I have met in practice.

ADDITIONAL READING

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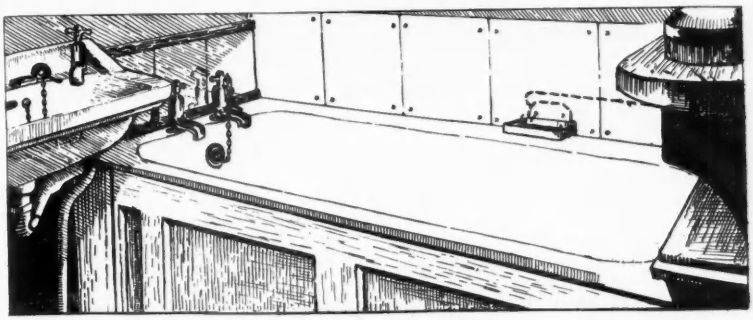

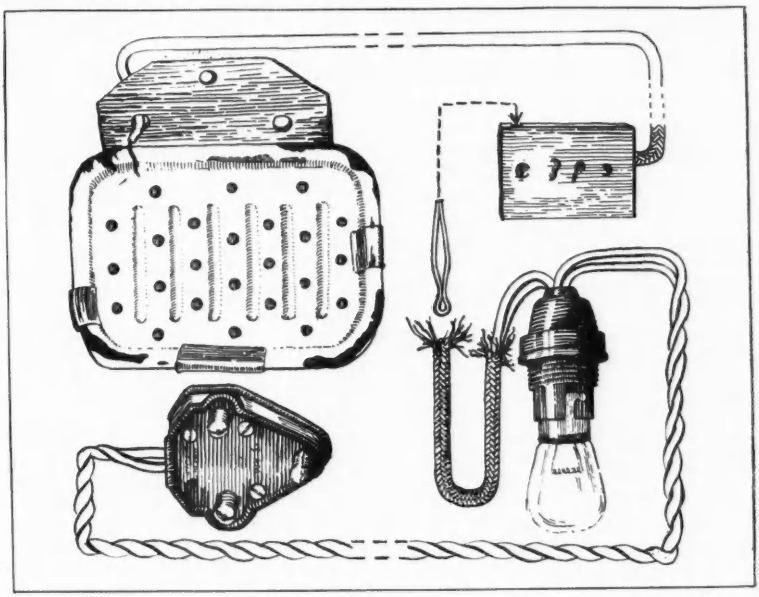

FIG. 2

Above: Bath showing soap dish (with wire indicated by dotted line).
Below: Soap dish and connexions to plug in bedroom.

TABLE I

ACCIDENT DEATHS FROM ELECTRIC CURRENT

	1942	1943	1944	1945	1946	1947	1948	1949	1950	1951	1952	1953
Male	88	94	79	81	90	78	77	68	69	85	94	86
Female	14	20	22	29	16	16	19	21	20	17	18	25

TABLE II

ACCIDENT DEATHS DUE TO ELECTRIC CURRENT
1952–53

	Total		*At work*		*At home*	
	M.	F.	M.	F.	M.	F.
1952	94	18	58	1	27	15
1953	86	25	56	3	13	22
	180	43	114	4	40	37

SPACE MEDICINE*

BY

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It may be felt that a discussion of this subject is a trifle premature at this time. However it is of interest to quote the words of Major-General H. G. Armstrong, U.S.A.F.,[1] referring to a similar discussion in 1952.

"The skill and ingenuity of the aeronautical engineer in designing aircraft with increased speed, endurance, and altitude performance, have currently projected the flying personnel into the immediate vicinity of an environmental frontier beyond which our knowledge is incomplete, and, in some respects, totally lacking. These gaps in our knowledge must be filled in at the earliest opportunity for at least the following reasons. A modern, high-performance military aircraft cannot be properly designed until the effect on the crew of the performance of that airplane and the environment through which it will fly is definitely known, because aircrew protective devices must be built-in design features of such an aircraft.

"This consideration points out a fact not generally recognized: that Aviation Medical Research, in this area at least, should be completed prior to the time the aeronautical engineer first sits down to his drawing-board, which is usually five to ten years before the projected airplane is first flown."

This illustrates the important and exciting part that medical science has to play in the conquest of space.

Over the past few years advances in science have brought the possibility of space travel within our grasp. However, there is little possibility of a space ship being able to make a successful trip without a human pilot. No computer which can cope with the multitude of problems liable to arise during take off and landing has yet been constructed; and it is quite impossible that one small enough to do this work could be built in the immediate future. Therefore, if we are to be successful, the space-ship will be manned by human beings. It is my purpose to point out to you some of the medical problems that will be encountered by the first voyager across the space frontier.

Firstly we have to face the fact that the human body was designed to operate efficiently on the surface of this earth. Obviously the putting of it into an unfamiliar environment will raise many problems. These may best be considered as they arise in a hypothetical "Journey into Space".

Before we "take off" I would like to clarify the position as regards the numerous so-called "barriers" that are frequently discussed in the popular press. By these I mean the so-called "sound", "heat" and "brain" barriers.

These are as realistic as the old idea that the human body could not travel faster than a hundred miles an hour. The only barriers that exist are due to gaps in our knowledge of conditions prevailing at various speeds. None of these need interfere in any way with our progression into space. Remember speed itself has no effect whatsoever on the human body; in fact all of us are travelling at a speed of a thousand miles per hour with the Earth's rotation! It is the change of speed, i.e. acceleration or deceleration, that brings the force of gravity to bear on the body and produces what are known as "*G* effects".

Now the "take off". In order to achieve 15,000 m.p.h., which is the minimum escape velocity, it will be necessary to accelerate very rapidly because of the rapid burning

* This is a paper presented to the Cornwall Clinical Society on 6th January, 1956.

of rocket fuel. This will inevitably produce a high-*G* loading on the pilot. This effect has been studied by using machines such as the man-carrying centrifuge, which simulates these high-*G* conditions. (There is one of these machines at the I.A.M. Farnborough and several in the U.S.A.). From these studies it is apparent that the effects of *G* on the body are dangerous mainly by their interference with the circulatory system. As the blood becomes heavier the load on the heart becomes greater and the circulation slows. The greater the *G* loading the slower the circulation becomes and pooling of blood in certain areas occurs. These are particularly the leg veins and abdominal veins. This pooling results in there being insufficient blood actually circulating. The amount of blood pooling is influenced by the position of the body relative to the line of resultant forces caused by change of velocity. Once the circulation has become greatly slowed, the brain, being the organ most sensitive to oxygen-lack, begins to stop working and dimness of vision, known as "grey out" and later loss of consciousness known as "black out" occurs. Another important factor is the extreme difficulty of movement under high-*G* conditions.

Our first step then will be to minimize the effects of *G*. This can be done in several ways, firstly by wearing a specially designed anti-*G* suit. This suit exerts its effect by pressing on the areas where blood pooling occurs and thus preventing this. It is automatically inflated when the *G* loading reaches a certain predetermined figure. This figure being found by testing beforehand in the centrifuge; and is the point at which the subject begins to notice visual or other disturbance. Secondly, the prone position is found to aid the circulation by reducing the height the blood has to be lifted, and increasing the *G* tolerance. Thirdly, the instruments will have to be power-operated, and very near the hands of the pilot. Fourthly the peak of physical fitness is essential.

Once the rocket has taken off it is necessary to provide a suitable atmosphere to breathe. The cabin will have to be pressurized to normal atmospheric conditions. The possibility of producing an artificial atmosphere by means of grasses in a so-called "botanical exchange unit" will not at present be possible, but may be essential later for longer trips. This would work because of the natural tendency of plants to absorb CO_2 and give off O_2; when irradiated with ultra-violet rays. Until more work has been done on this it seems likely that stored O_2 will have to be used. The pilot will not be wearing a space suit as this will be far too cumbersome for the wearer to carry out any intricate tasks. In fact he may be wearing nothing at all (in the absence of lady members of the crew) as there will be at first a considerable rise in the internal temperature of the cabin due to friction effects. (Additional active cooling plant may be necessary.)

What of the problem of a space suit? You may have seen pictures released by our American friends of a high-altitude or space suit designed for the U.S. Navy. Knowing their efficient security methods, you can be sure it is valueless! Otherwise such a detailed picture would not have been released. The problem is not so much to produce a suit which would be fully pressurized, as to produce one in which the individual can move about. A rigid suit could be constructed now, but would, of course, be useless. However, this is mainly an engineering problem and advances in flexible, tough materials are bringing the solution rapidly nearer.

Having reached the escape velocity we will begin to leave the region of the earth's gravitational field. The effect of gravity will rapidly decrease, as the force of gravity obeys the inverse square law of distance. Therefore we shall soon be under conditions of low or absent gravity and this will cause many physiological problems. The labyrinthine apparatus which normally regulates our sense of position and balance will start to present us with equivocal information, and it is possible that extreme nausea, vomiting, and vertigo may occur (as in labyrinthine disease). Also this will mean that to maintain orientation vision will be essential. In addition the blood pressure and speed of circulation will increase if the heart continues at its ordinary rate, for the speed of circulation is influenced by the stagnation effects in the veins produced by gravity.

Both these effects may be very serious and it seems likely that heavy medication will be necessary to slow the heart and to overcome the vertigo, &c.

Another serious problem will be eating and drinking. Drinking will be difficult because in the absence of gravity, fluid will not pour out of its container, and swallowing of solids is difficult because again there is no gravity to carry food to the back of the throat where the muscles of deglutition can take effect. Possible ways of overcoming this are by having both food and drink in capsules, or sucking through a tube.

Further, this lack of gravity will produce difficulty in eliminating faeces and urine, and extreme care will be necessary to avoid the spilling of them in the cabin! The possibility of having the space rocket spinning to provide a centrifugal "gravity" should not be considered likely at present, as it adds too greatly to the problem of controlling the rocket.

As soon as we leave the shielding effect of the atmosphere we shall be exposed to cosmic radiation. Much speculation has occurred about the dangers of cosmic rays. Now what is cosmic radiation? Mainly it consists of atomic nuclei freed from their surrounding electron rings.

These nuclei have only small powers of penetration of solids and it seems likely that the metallic space-ship walls will provide sufficient protection from the majority of them. However, there is a secondary danger because on collision with the metallic walls these nuclei will seize electrons from the atoms in the wall molecules, with the resultant release of small quantities of gamma radiation. Gamma radiation has great penetrating powers and is responsible for some of the dangerous effects of an atomic explosion; therefore if the quantities of gamma radiation are high it is likely that this effect will be extremely dangerous. Rocket experiments so far show that at a height of 200 miles this radiation is not serious and no serious genetic changes have been detected with prolonged similar doses in animal experiments.

What now of the psychological aspects? In every mind there dwells the elements of both claustrophobia and agorophobia. These will be magnified greatly by this voyage into the unknown. The cabin will be very small and there probably will be no direct vision ports, vision being achieved by radar and by periscope. When the pilot does look out through the periscope the vision of space as seen from outside the Earth's atmosphere is almost impossible to imagine! It is easy to say that the sky will be black and the stars bright; but the great impression will be the vastness of the universe, and the utter insignificance of man and his works in relation to this.

The question of so-called space sickness is at present undetermined. There seems no reason why it should occur. From our present knowledge of car, sea, and air sickness it seems that the factors are:

(1) a susceptible individual,

(2) unaccustomed motion,

(3) psychological factors varying from individual to individual.

In the absence of a susceptible individual there seems no way of producing motion sickness. Even bringing about complete disorientation by whirling a person round and round for some time cannot produce motion sickness in a resistant person. Pretesting to extreme limits would be essential before selecting the pilot.

In addition to these known syndromes, experimental work has indicated the possibility of a new syndrome brought about by interference with three of the basic facts to which the mind and body are accustomed:

(1) The instinct of survival, there being no escape from the rocket,

(2) The removal of the earth which the mind has always accepted as being near,

(3) Absence of gravity which the body has always experienced.

It may be necessary to provide hypnotic conditioning, and post-hypnotic commands may have to be instilled in the mind before it can accept these multiple changes of environment.

The final question is what is the danger of collision with a meteorite? Although this

is not a medical question I would like to emphasize that calculations taking into account the relative size of the rocket, and the relative frequency of meteorites as counted by the radio-telescope, have shown this to be only a small danger.

So we are left with the thought that some day the vastness and splendour of the Universe may unfold before us like a beautiful garden, waiting to be explored by an enlightened race of men.

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[1]Armstrong, H. G. (1952). *Physics and Medicine of the Upper Atmosphere* (Foreword). University of New Mexico Press.

HYPERCALCAEMIA WITH HYPOCALCURIA
AN UNUSUAL CASE OF PRIMARY HYPERPARATHYROIDISM

BY

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The importance of this case lies in its great rarity, and the presence of hypercalcaemia with a low urinary output of calcium in hyperparathyroidism due to parathyroid adenoma which was proven by autopsy. Only three other similar cases have been described.

THE CASE-HISTORY

Miss Ellen H., aged 74, had had rheumatoid arthritis which began in her thirties, but had not been troublesome for many years. Aged 39 she knocked her right breast in which a tender lump developed, and the whole breast grew larger than the other. For this condition she had a radical mastectomy at the age of 45.

She was in good health until, four years before admission, spinal curvature gradually developed, and she noticed weakness first in the right leg and later in both. The weakness and bowing of the spine increased steadily. One year before admission intermittent aching pains in the back and legs began and grew worse. Six months later she became bedridden because of the weakness of her legs. Control of the sphincters remained normal. She lost flesh and the spinal deformity and pains increased in severity.

Mentally unclouded, and suffering her disabilities with fortitude, she was, like her siblings, only about five feet tall, and had a severe kyphoscoliosis. Her ribs were collapsed inwards narrowing the chest from side to side, and the sternum and clavicles were arched forwards. Her head seemed disproportionately large, but was 21½ inches in circumference. The thyroid gland was not enlarged, and no other abnormal mass was palpable in the neck. There was a generalized wasting of muscle and fat. Her legs were paralysed with increased tendon reflexes, extensor plantar responses and sustained ankle clonus. A bony lump was palpable on the left shin. Her upper limbs were weak and spastic. Her hands showed evidence of burnt-out rheumatoid arthritis, with enlarged joints and ulnar deviation of the fingers. Both radii were curved with the concavity forwards. No abnormality was found in the abdomen, respiratory and cardiovascular systems. The arteries were supple and the blood pressure 145/90 mm. Hg. At no time was albumen found in the urine whose specific gravity ranged from 1006–1020.

The following investigations were done:

Serum Calcium	14·9 mg. per 100 c.c.
Serum Inorganic Phosphorus	2·7 mg. per 100 c.c.
Serum Alkaline Phosphatase (K.A. Units)	60 units per 100 c.c.
Serum Albumen	3·7 g. per 100 c.c.
Serum Globulin	2·1 g.
Thymol Turbidity Test	2·7 units.
Colloidal Gold Test	1 +

Blood Urea	58 mg. per 100 c.c.
W.R. and Kahn Reactions	Negative.
Serum Calcium	14·3 m.g.
Serum Ionic Calcium	7·5 mg.
Urea Concentration Test	12% of normal.
Plasma Chlorides	573 mg. per 100 c.c.
Plasma CO_2 Capacity	64 vols.

On three occasions in one week Barney and Sulkowitch's Test gave the faintest cloud indicating Calcium excretion within normal limits.

Radiographs of the bones showed generalized decalcification. A few areas appeared normal, but the whole skeleton was affected, either by abnormal curving, or by scattered rarefied areas due to osteoclastomata, and by thickening or thinning of the bony cortex. Most of the large bones showed all these features. (Plate VIII (*a*) and (*b*).)

The urinary calcium excretion was now studied (see Table I). The patient was given a low-calcium diet which suited her palate. She kept rigidly to it from 10th August to 7th September. A day's supply of food was ashed, and contained 74 mg. of calcium and 234 mg. of phosphorus. She drank exactly 30 oz. of fluid a day until 27th August 1951 when she was urged to drink as much as she could to see if more calcium could be excreted. As the water in Birmingham is very soft calcium intake from this source was negligible.

TABLE I

Date	Fluid Intake in oz.	Fluid Output in c.c.	24 hr. Urinary Excretion		Serum		
			Calcium mg./day	Phosphorus mg./day	Calcium mg.%	Phosphorus mg.%	Alkaline Phosphatase K.A. Units per 100 ml.
Aug.							
9th	—	—	—	—	14·2	3	—
16th	30	840	40	215	—	—	—
17th	30	890	55	332	15·3	2·6	52
24th	30	800	41	—	—	—	—
27th	41	—	—	—	—	—	—
28th	39	—	—	—	—	—	—
29th	41	—	—	—	—	—	—
30th	87	—	—	—	—	—	—
31st	67	—	—	—	—	—	—
Sept.							
1st	41	1260	61	—	—	—	—
2nd	53	—	—	—	—	—	—
3rd	49	—	—	—	—	—	—
4th	82	1100	64	—	—	—	—
5th	—	—	—	—	—	—	—
6th	51	—	—	—	—	—	—
7th	47	—	—	—	14·9	2·2	55

There was a steady increase of her disabilities. First one then the other femoral vein thrombosed, and she grew weaker and more bent. She died quietly and unexpectedly.

THE AUTOPSY FINDINGS

The examination, 53 hours after death, revealed a marked porosis of the bones which cut with a saw like rotten wood. Both femora showed moderate thickening of the cortex and dilation of the shafts in the upper thirds by copious red marrow tissue. The calvarium was thickened and could be cut by a knife with moderate pressure. The brain, apart from two areas of old symmetrical softening in the basal ganglia, and

PLATE VIII (*a*)

This radiograph of the right humerus, chest, and spine illustrates the bowing of the bones (spine clavicle and scapula), the variation in thickness of the cortex, and the osteoclastomata in the head and shaft of the humerus and tip of the scapula.

PLATE VIII (*b*)

The lateral radiograph of the skull, showing the thickened cortex, and mottling due to numerous small osteoclastomata. Its appearance could be mistaken for that of Paget's disease.

moderate atheroma of the cerebral arteries, was of normal appearance. The thyroid gland had a small adenoma in the upper pole of the right lobe, but was otherwise normal. On the lower lateral aspect of its right lobe was a soft capsulated tumour measuring $3 \times 2\frac{1}{2} \times 2\frac{1}{2}$ cm. The cut surface of this tumour was a deep brownish red colour. No other parathyroid glands were found. The lungs were emphysematous and showed a confluent bronchopneumonia in the right lower lobe. There was atheroma of the coronary arteries and aorta but the myocardium, apart from a few minute scars in the left ventricle was normal.

The liver, spleen, pancreas and suprarenals were normal. Both kidneys were reduced in size and weighed 142 g. The surface was granular. The cortices were narrow and not well differentiated from the medullae. There were several minute, finely granular patches of metastatic calcification on the cut surfaces. The rest of the urinary tract and the other organs of the body were normal.

Sections showed the parathyroid adenoma to have an acinar structure supported by a network of capillaries. The cells were polygonal with finely vacuolated and slightly acidophil cytoplasm and round deeply stained nuclei. Other parts did not have any particular arrangement and the cells were round with watery, slightly basophil cytoplasm, and distinct round or slightly oval nuclei. There were also a few minute islets of small oxyphil cells with pinkish cytoplasm. Kariokinetic figures were found in one or two cells only. Very occasionally some of the ballooned principal cells had two nuclei.

Sections of the kidneys showed thickening of the arteries, and fibrosis of some glomeruli with slight surrounding infiltration by small lymphocytes. There were scattered areas of calcium deposits mainly confined to the medulla. Sections of the stomach, lungs, suprarenals, pancreas, and heart did not show any metastatic calcification.

In the femur there was great osteoclastic activity. The cortex was reduced to slender trabeculae embedded in fibrous marrow, which contained nodules of osteoclastomata. There was no evidence of delay in calcification such as occurs in vitamin D deficiency.

There was no evidence of an attempt to restore new bone. In the sections of bone examined there was nothing to suggest the presence of Paget's disease.

DISCUSSION

In primary hyperparathyroidism there is usually one adenoma, rarely more, and occasionally hyperplasia of all four glands. The excretion of parathormone by this neoplasia (whether fulminating, steadily progressive, or fluctuating), the calcium intake, and the presence of other diseases, determine the mode of presentation and the course of the disorder.

If the adenoma produces rapidly increasing amounts of parathyroid hormone the patient will die suddenly with great muscular weakness, and anuria (Dawson and Struthers, 1923), but this is exceedingly rare.

Usually the disease runs a fairly steady course, and if it presents before the renal lesions have become severe, the way in which it does so probably depends upon the calcium intake of the patient, who, if he likes milk will develop urinary stones (Albright and Bauer, 1934). If he has a low calcium intake the bony softening will predominate, with pain, deformity, fractures, and ultimately paralysis from spinal cord compression. In all cases calcium phosphate is deposited in the tubules and its presence leads to fibrosis with destruction of the kidney. Subjects with severe renal calcinosis may present with uraemia, and an example of this is reported by Downs and Scott, 1941. In life this patient's serum calcium was low and phosphorus high, and he had a low urinary excretion of calcium. At autopsy an adenoma of one, and hyperplasia of the remaining three parathyroid glands was found. The kidneys had gross calcinosis and fibrosis, but the bones were little affected. It is considered that the adenoma was the initial lesion, and caused the calcinosis, which eventually produced renal failure which in turn induced the other parathyoid glands to hypertrophy. (Ham 1940, Snapper 1949.)

It is possible that some of the few cases reported of patients with proven parathyroid adenomata and bony decalcification in whom the urinary calcium excretion is low may be progressing to the same end as the case mentioned above. A low or normal urinary calcium excretion in the presence of hypercalcaemia may seem incongruous, but it has previously been reported in five cases. Two of these had parathyroid adenomata (Snapper 1949, and Gutman and Parsons 1938, Case 2), and one hyperplasia of all glands in which the main cell was of a type found usually in secondary hyperplasia, though the authors doubt that the kidney lesion could have been primary (Pratt, Green and Neuhauser 1947). The other two cases were sisters with renal rickets, one of which was shown to have renal calcinosis and fibrosis at autopsy (Chown 1936). Two cases of Albright's *et al.* (1934) with proven parathyroid adenomata and poor renal function tests had urinary excretions of calcium which though raised were much lower than usual in hyperparathyroidism. These cases had a greater faecal calcium excretion than normal. The authors suggest that this may be compensatory in cases where renal failure reduces calcium excretion in the urine. The phenomenon of hypocalciuria with hypercalcaemia, is rare, and the mechanism unknown, but a characteristic of the three cases who came to post-mortem examination, or who had renal function tests done, was severe kidney failure, and this might provide a simple *a priori* explanation. It should be pointed out however that many cases of primary hyperparathyroidism with comparably bad renal function tests do excrete excessive amounts of calcium. Snapper 1949 explains the phenomenon by postulating a vitamin D deficiency, common in northern China where he observed his case. Since in the osteomalacia of this region a very low urinary calcium has been observed when the blood calcium was normal (Liu *et al.*, 1940), he suggests that in hypovitaminosis D and hypercalcaemia from hyperparathyroidism, low excretion of calcium in the urine might occur. Adequate renal function tests were not reported in this case history. This type of osteomalacia has not been seen outside northern China (Albright and Reifenstein, 1948). It is therefore not a likely explanation.

The disease may run a fluctuating course (Willich, 1920, and Linden, 1934) and this provides an explanation of some of the cases in which one bone may have the appearance of Paget's disease on X-ray (Albright, 1934, and Gutman and Parsons, 1938) while the remainder of the skeleton is typical of hyperparathyroidism. (See Plate VIII (*b*).) Recalcification in periods of normality could produce the appearances described. Gutman and Parsons (1938) do, however, report a case in whom the two diseases were probably present together, for the alkaline phosphatase remained high for two years after the adenoma had been removed, which is unusual. Albright and Reifenstein (1948) have seen others.

The case presented is of proven primary parathyroid adenoma with hypercalcaemia and a consistently low urinary calcium excretion. In life her kidney function tests showed poor function and at autopsy there was severe renal fibrosis from calcinosis. She fits with the other cases quoted above into the natural history of hyperparathyroidism illustrated in Table II. The present case and those culled from the literature show that though a high urinary excretion of calcium is induced by excessive production of parathormone, this may not always occur in cases of hyperparathyroidism with severe renal defect, and should not be used as a rigid diagnostic criterion when the latter is present.

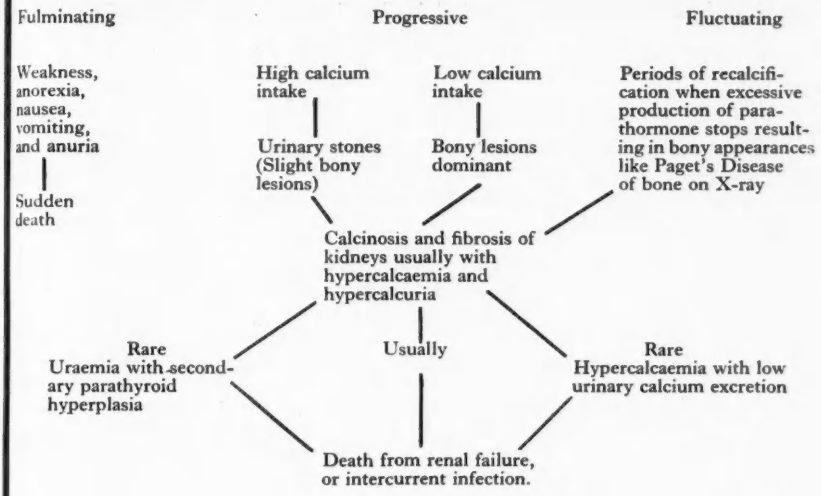
SUMMARY

The natural history of primary hyperparathyroidism is discussed, and a case of primary parathyroid adenoma with hypercalcaemia but low urinary calcium excretion is described.

Our thanks are due to Mr. R. P. S. Kelman and Dr. G. G. Gillam of Selly Oak Hospital, Birmingham for permission to publish this case.

TABLE II

THE NATURAL HISTORY OF PRIMARY HYPERPARATHYROIDISM


Fulminating
Progressive
Fluctuating
Weakness, anorexia, nausea, vomiting, and anuria
Sudden death
High calcium intake
Urinary stones (Slight bony lesions)
Low calcium intake
Bony lesions dominant
Periods of recalcification when excessive production of parathormone stops resulting in bony appearances like Paget's Disease of bone on X-ray
Calcinosis and fibrosis of kidneys usually with hypercalcaemia and hypercalcuria
Rare
Uraemia with secondary parathyroid hyperplasia
Usually
Rare
Hypercalcaemia with low urinary calcium excretion
Death from renal failure, or intercurrent infection.

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GENERALIZED OSTEITIS FIBROSA WITH PARATHYROID ADENOMA

(A Clinical Pathological Conference of the University of Bristol Medical School.)

CHAIRMAN: PROFESSOR T. F. HEWER

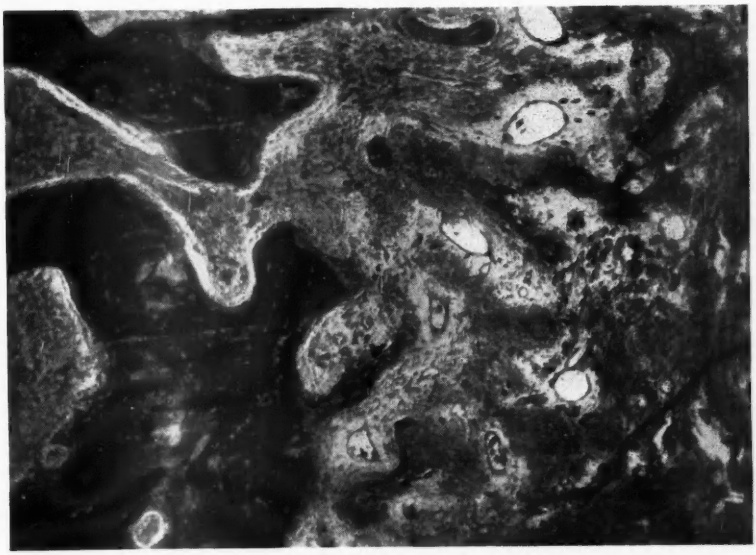
Dr. J. M. Naish: I first saw this patient in Snowdon Road Hospital. As she was only forty-five and therefore much younger than is usual for patients in this hospital I thought as I approached the bed that she had some interesting neurological condition or disseminated sclerosis but I was told that she had had some pain around the groin, that her doctor had sent her for X-ray of the pelvis, and that a large destructive lesion had been found in one of the pubic rami. A surgeon saw her and advised that she should be admitted to a chronic hospital: the diagnosis was secondary tumours of bone. When I first looked at this X-ray picture of the pubis and one of the skull, which was taken because she had exophthalmos of one eye, I fell for the diagnosis and looked for a primary, but I was struck by her good general condition. There was no obvious primary; the thyroid and breasts were normal. The uterus had been removed some years previously. The kidneys were just palpable—we had a pyelogram done and that was normal. I felt it was probably not a malignant condition. When she could walk properly we let her go home and I saw her in out-patients' on two occasions and then readmitted her to Southmead. At that time the condition which was running through my mind was myelomatosis or xanthomatosis: I suggested that a biopsy should be done and Dr. Brown had the specimen. He gave a most interesting report, as a result of which we had the serum calcium and phosphate levels estimated and they were grossly abnormal: serum alkaline phosphatase 58 units per 100 ml., serum inorganic phosphate 2·5 mg. per cent., serum calcium 14·4 mg. per cent. These were so abnormal that they entirely confirmed Dr. Brown's suggestion and as X-ray pictures were also confirmatory we sent her to the surgeon to find the tumour which we could not feel!

Mr. A. G. McPherson: My job was a straightforward one; I had to look for a parathyroid tumour. She was pretty thin; it was a scraggy neck and there was nothing to feel in it. I did a standard thyroid incision and exposed the whole thyroid by dividing across the strap muscles. I started on the left lobe, looked at all aspects, divided both main arteries and finding nothing I divided the pre-vertebral fascia and found nothing except one or two little nodules on the thyroid. I went to the right side and again I turned the lobe practically over, examining the whole of the back and could feel nothing. I then divided the fascia over the pre-vertebral muscles, slipped my finger down there and then I felt something; it was in front of my finger, and after that it was relatively easy to dissect it out. (Two photographs taken during the operation were then shown.)

Dr. N. J. Brown: Let us first consider the bone biopsy. The histological changes are well illustrated by the slides. (See Plate IX (*a*).) The principal features are erosion of the bone and replacement by fibrous tissue. In places (Plate IX (*b*)) there are clusters of osteoclasts nibbling away the bone. There is very little evidence of cyst formation. It is clearly a form of fibrous dysplasia of bone and Dr. Lewis and I both felt that there was a strong possibility of this being a case of generalized osteitis fibrosa cystica, due to overactivity of the parathyroids. We sent out a report to this effect and suggested further investigation along these lines.

Now to turn to the parathyroid tumour which Mr. McPherson removed. This measured 4×2×2 cm. and was mainly solid with a few small cysts. Histologically (Plate X (*a*)) it shows a uniform picture, being composed of small cells with regular round nuclei. It is a simple parathyroid adenoma composed of "chief" cells.

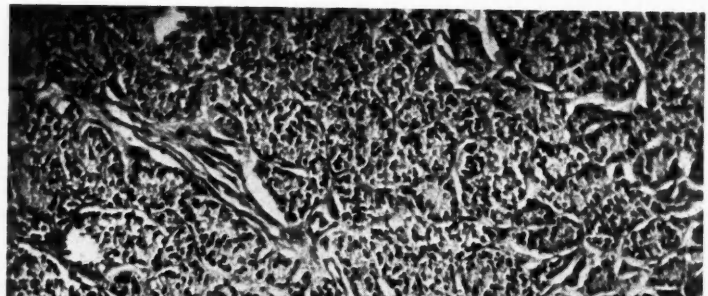
Dr. G. R. Airth: My first contact with this patient was a request from her family doctor for examination of the right hip joint; a chest film had been taken some days



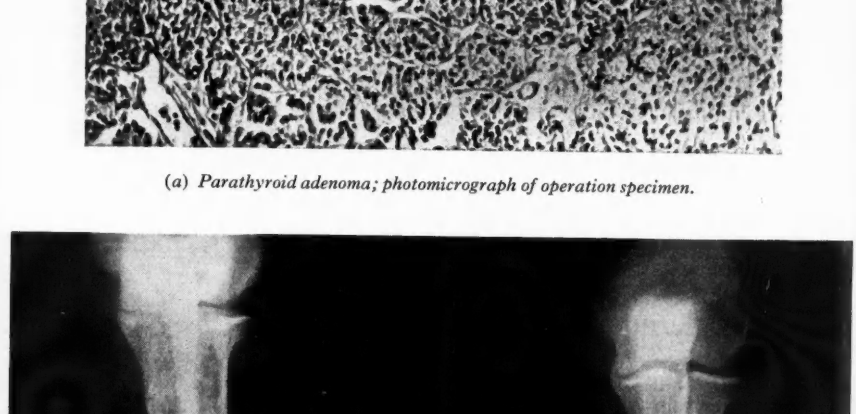
(*a*) *Biopsy of pubis: erosion of bone and replacement by fibrous tissue.*

(*b*) *Biopsy of pubis: osteoclastic resorption of bone.*

PLATE X



(*a*) *Parathyroid adenoma; photomicrograph of operation specimen.*



(A) (B)

(*b*) *Radiographs of radius and ulna:* (*A*) *shortly before operation and* (*B*) *nine weeks later.*

previously. An extensive lesion of the symphysis pubis was present and others involved the right acetabular floor and the left ischial ramus. I received vague information from the patient that she had had "half her inside out" and was guilty of calling the bone lesions metastases on these thin premises. I advised reference to a physician. Later I learned that she had also been seen at the Bristol General Hospital, where similar conclusions were drawn. Six weeks later I saw films of skull and ribs; the multiple lesions still suggested metastases. In the search for a primary lesion producing osteolytic metastases, pyelograms showed no evidence of a renal lesion. The patient appeared again, nearly seven months after the hip episode, and it was clear that in spite of new lesions, and enlargement of the old, the cortex was being pushed aside and not being destroyed; myelomatosis was suggested. Finally, when biopsy and biochemical findings were known, the most appropriate bones were examined for the first time; the radius (Plate IX (*b*)) showed severe osteoporosis, with absorption of compact bone and a cystic space in its middle third. The metacarpals and phalangeal shafts showed many small cysts. Spontaneous fractures were shown where the cystic spaces occupied much of the bone thickness, as in the case of the ribs and ischium.

Dr. Naish: Ten days after the operation she fractured her femur—apart from that she had an uneventful recovery.

Dr. Airth: Nine weeks after the removal of the parathyroid tumour the healing process was already well advanced (Plate X (*b*)); the skull looked entirely normal, the pubic lesion was shrunken and sclerosed, the cysts had entirely disappeared, and in the case of the radius the only abnormality was a small midshaft area of sclerosis.

Dr. F. J. W. Lewis: The laboratory findings confirmed the diagnosis. There was a very high alkaline phosphatase, a raised plasma calcium and lowered inorganic phosphate. Hyperparathyroidism causes increased excretion of phosphate and therefore lowers the serum phosphate throwing out the calcium phosphate balance and thus causing a rise in calcium. On the findings obtained I felt quite confident of our diagnosis.

Dr. J. E. Cates: Many parathyroid tumours are not associated with decalcification; this has been noticed particularly in the North American continent where the dietary intake of calcium is high. In these cases the phosphatase may be quite normal, but the serum calcium is high and quite often there are renal stones. Another interesting feature of the raised calcium level is the mental changes; the patients become depressed and mentally slow. It would also be interesting to know whether this patient had any signs of tetany when the tumour was taken out.

Dr. Naish: This is a very rare condition and the only case I have seen myself. With regard to the renal complications; they can be very serious. I do not know anything about the patient's diet—I think it was quite normal. About her mental changes: she was an exceptionally nice person and I think she probably felt very depressed and miserable but she never showed unpleasant traits or behaviour. About getting post-operative tetany: we tried to guard against this by giving her additional calcium four days before the operation and two or three weeks after. We had no post-operative complications.

Mr. R. V. Cooke: I can speak of quite a few cases: one of the two most interesting and instructive ones was one from Southmead Hospital about twenty years ago: there was in Southmead a sort of dwarf—a little boy who had had many stones removed from his kidney and he had a very high blood calcium—18·8—and we found this parathyroid tumour. The strange thing was that he had been regarded as a mental case—almost an idiot—and rather the joke of the hospital. He had the tumour removed about the first week in December and by Christmas he was the wag of the ward! He became bright and intelligent and good fun: quite a different picture.

The next case was one which was missed and I suppose every excuse was found for missing it! This was a patient who was in the General Hospital about fifteen years ago with a large swelling in her face; she had a tumour removed and it was a big clean cavity when I saw it. Fortunately for her, just before she left hospital, she developed

a swelling in the upper end of her tibia which was visible and the surgeon asked me to see her. I saw her and the hole in her cheek and the lump in the tibia and I said to the students "Now what is the diagnosis?" They said it was very hard and I said that there were many suggestions we might make. They thought secondary malignant metastases from this tumour in the upper jaw. She was a young woman and she was pretty fit physically but dull mentally. I said, "I think this is a parathyroid tumour and what is more, here it is!" When she swallowed, you could feel this tumour coming out of the superior thoracic border and it was quite easy to feel. So here was the whole clinical picture—no need for any tests. It was so unusual that Professor Perry, to whom I referred the case, thought it was a figment of my imagination but I am not an imaginative person and I said, "Well—there it is." We took pictures of other bones, the long bones, and there were many cystic spaces. She was removed from the surgical ward and fractured her femur on the way to the medical ward. We operated on her: I made a tiny little incision about two inches long and removed the tumour: that was the whole operation and it took about a quarter of an hour. She got well and remained well for a number of years. After about seven years, during which time her blood chemistry was reviewed from time to time in the out-patients' department, things started to go wrong again. Her calcium went up and the condition was recurring: it was not a persistent case but a recurring case. She began to get cysts again and she fractured another long bone. Her mental condition became quite definitely better after the first operation; she became quite charming. When we explored her the second time, I examined the thyroid thoroughly and found nothing very dramatic in the way of a parathyroid tumour. I did eventually find a small nodule about 1 cm. in diameter which was different in colour from the rest of the thyroid, then I found another and another—there were three. Since she had a nodular thyroid, it was difficult to say these were anything more than nodular degeneration of the thyroid, so I sent these nodules to the pathologists. In due course their report came through—they were all three parathyroid tumours. So her blood calcium came down again, she began to get signs of tetany which were not difficult to control and she got better again and she is still in Professor Perry's wards. We learn from this case that you may have more than one parathyroid tumour and this possibility must be remembered in the case under discussion this evening. I would like to ask what is a normal figure in the blood calcium and what should be regarded as abnormal? If we are looking for evidence of a tumour, what figure should we be suspicious of? I should have thought 11 was above the normal or even 10·6 mg. per cent.

Dr. Lewis: It averages about 10 but I think you would be on dangerous ground if you act on the assumption that 11 mg. per cent. is abnormal.

Dr. Airth: How would Mr. Cooke have diagnosed the finding on the X-ray pictures in this evening's case?

Mr. Cooke: In the absence of any other explanation, hyperparathyroidism is worth thinking about.

Notes and News

Dr. Joseph Harold Sheldon received the honorary degree of Doctor of Medicine on July 3rd, 1956. Dr. Sheldon's best known works are his monographs "Haemachromatosis" and "Social Medicine of Old Age". This honorary degree was conferred last in 1938 on Sir William George Savage.

Mr. A. L. Eyre-Brook, on the invitation of the Secretary of State for the Colonies, is on a ten week visit to the Western Pacific territories as Consultant Adviser to the Colonial Medical Service.

Dr. T. E. Oppé, M.R.C.P., D.C.H., has been appointed Lecturer in Child Health.

Dr. D. Bowden has been appointed Assistant Professor of Pathology at St. Louis University, Missouri.

Dr. David W. Pugh and Dr. J. E. Cates have been elected Fellows of the Royal College of Physicians.

UNIVERSITY OF BRISTOL

Doctor of Medicine, Honoris Causa, Dr. Joseph Harold Sheldon.

M.B., Ch.B. (*June*, 1956)

WITH SECOND-CLASS HONOURS

Pauline Frances Haswell, (with distinction in Public Health).
Dennis Howard Wright.

PASS

Miriam Mary Alderwick.
Babatunde Oladipo Amure.
Elizabeth Margaret Forsythe Andrews.
John Henry Andrews.
John Christopher Bailey.
Edwin Peter Beck.
William Edward Benny, (with distinction in Public Health).
Brian Thomas Allaway Clarke.
Ruth Ethel Ashurst Coles.
Harry Edward Cramp.
John David Davison.
Keith Morton Evans.
Irene Elizabeth Gibbs.
Paul Michael Gill.
Claude Louis Godfrey Hale.
Arthur Frederick Hickson.
Alex Leonard Horton.
Rachel Mary Hulbert.
Alan Thomas Lloyd.
Sheila Margaret McClements.
June Margaret McLeod.
Terence Morley.
Richard Walford Morris.
Anezionwu Nwankwo Okoro.
Kenneth Michael Parry.
Weekakkodige Fredrick Perera.
Clement Keith Protheroe.
Michael John Rose.
William John Stephen.
Stuart James Strong.
Jeremy Philip Telling.
Michael James Watson.
Anthony Harold Elvin Williams.
Mary Bowen Wilson.
Christopher Holman Wood.

PRIZES

Edward Fawcett Memorial Prize for 1956: Miss S. J. Smith.

The Foyle Prize for 1956: Miss M. J. Smith.

Richard Clarke Prize in Child Health: Mr. D. H. Wright.

Russell Cooper Prize: Mr. D. E. Bowden.

The Thomas Edgeworth and Francis Henry Edgeworth Prize in Physiology: Mr. J. R. Winslade.

Tibbits Memorial Prize for 1956: Mr. D. H. Wright.

William Hyde Award (£300 p.a.): Dr. Martin Herford.

Given by Research Board for the Correlation of Medical Science and Physical Educational Studies of mental and physical problems of young people leaving school and entering industry.

EXAMINATION RESULTS

M.R.C.P. (*London*). J. S. Bearcroft, M. S. Dunnill.

Appointments

UNITED BRISTOL HOSPITALS

MARCH—MAY, 1956

Name	*Appointment*	*Formerly*
ROBERTS, DOUGLAS IVOR, M.B., B.S., F.R.C.S.(Eng.)	Senior Surgical Registrar	Surgical Registrar, U.C.H., and Clinical Assistant, St. Paul's Hospital.
FREEMAN, ANTHONY GEORGE, M.A., M.D., M.R.C.P.	Senior Medical Registrar (exchange of appointment with Dr. Crow)	Senior Medical Registrar, Exeter Clinical Area.
YELL, JOAN, M.B., B.S., M.R.C.P.	Medical Registrar	Medical Registrar, Dryburn Hospital, Durham.
ALMS, MICHAEL, M.B., F.R.C.S., M.CH.ORTH.	Senior Orthopaedic Registrar	Orthopaedic Surgeon to Colony of Mauritius.
ROFFEY, PETER JAMES, M.B., B.CHIR., F.F.A.R.C.S., D.A.	Senior Anaesthetic Registrar	Anaesthetic Registrar, Hammersmith Hospital and Postgraduate Medical School.

SOUTH WESTERN REGIONAL HOSPITAL BOARD

BRISTOL

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SLEIGH, BEATRICE E., M.B., CH.B.(MANCHESTER–, D.A.(ENG)., F.F.A.R.C.S.	Senior Anaesthetic Registrar, Frenchay Hospital, Bristol.	Anaesthetic Registrar, Frenchay Hospital, Bristol.
CHANDLER, C. C. D., M.B., B.S.(LOND)., D.A., F.F.A.R.C.S.	Anaesthetic Registrar, Frenchay Hospital, Bristol.	Anaesthetic Registrar, United Bristol Hospitals.
ROBINSON, RUTH M., M.B., B.S.(LOND.), PRIMARY F.R.C.S.(ENG.).	Surgical Registrar, Weston-super-Mare General Hospital.	Locum tenens S.H.O. in Surgery at Weston-super-Mare General Hospital.
GALLA, T., M.B., CH.B. (EDIN.), D.P.M. (R.C.P. & S.)	Assistant Psychiatrist, Bristol Mental Hospitals.	Senior Registrar, Bristol Mental Hospitals.

NORTH GLOUCESTERSHIRE

Name	*Appointment*	*Formerly*
ENGLAND, E. J., M.B., B.S. (ADELAIDE), Primary F.R.C.S.(ENG.).	Surgical Registrar, Gloucestershire Royal Hospital, Gloucester	Locum tenens Surgical Registrar at same hospital.
READER, D. M., M.B., B.S. (LOND.), D.(OBST.)R.C.O.G.	Registrar in Obstetrics and Gynaecology, Gloucestershire Royal Hospital, Gloucester.	Gynaecological House Surgeon, Birmingham & Midland Hospital for Women.

BATH

Name	*Appointment*	*Formerly*
BERNSTEIN, CYRIL, M.A. (CAMBRIDGE), M.R.C.S., L.R.C.P.	Medical Registrar, Bath Group of Hospitals.	Locum tenens Medical Registrar St. Martin's Hospital, Bath.
WATT, P. A., M.A., M.B., B.CHIR., D.(OBST.) R.C.O.G.	Medical Registrar, Bath Group of Hospitals	Locum tenens Medical Registrar Royal United Hospital, Bath.
CONWAY, H. J., M.B., B.CH., B.A.O., D.P.M.(R.C.S.I.)	Assistant Psychiatrist, Roundway Hospital, Devizes.	Registrar, Parkside Hospital, Macclesfield.

SOUTH SOMERSET

Name	*Appointment*	*Formerly*
BARRY, D. M. R., B.A., M.B., B.CH., B.A.O., L.M. (ROTUNDA), D.M.R.(D.).	Consultant Radiologist, South Somerset Clinical Area.	Consultant Radiologist, Bolton Royal Infirmary

DEVON AND CORNWALL

Name	*Appointment*	*Formerly*
BURDON, J. F., M.B., B.S. (DUNELM).	General Practitioner Anaesthetist, Paignton Hospital.	In general practice, Paignton.
MANSFIELD, G. C., M.B., CH.B. (GLASGOW), D. (OBST.)R.C.O.G.	General Practitioner Anaesthetist, Paignton Hospital.	In general practice, Totnes.
TROBRIDGE, G. F., M.B., CH.B. (B'HAM), M.R.C.P. (EDIN.).	Assistant Chest Physician, Hawkmoor Chest Hospital.	Registrar, Hawkmoor Chest Hospital.
CANTRELL, G. L., M.B., CH.B. (MANCHESTER), D.O. (LONDON).	Consultant Ophthalmic Surgeon, Exeter Clinical Area.	Senior Registrar in Ophthalmology, United Bristol Hospitals.
CRAFT, M. J., M.B., B.S. (LOND.), M.R.C.P. (EDIN.)	Deputy Medical Superintendent, Royal Western Counties Instn. Starcross.	Registrar, Bethlem Royal & Maudsley Hospitals, London.
WEEKS, K. F., M.B., B.S. (LOND.), D.P.M.	Assistant Psychiatrist, Moorhaven Hospital, Ivybridge.	Senior Registrar, Warlingham Park Hospital.